Program and Abstracts

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Symptom Evolution Following the Emergence of Maize Streak Virus

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Abstract

When a pathogen evolves, it is expected that natural selection will modulate the severity of any pathogen-induced disease traits that either directly or indirectly impact its transmission probability. Accordingly, for many pathogens it has been demonstrated that the basic reproductive numbers are determined by evolutionary trade-offs between the pathogen-induced mortality rate, and the transmission rate. Given that in almost all cases these trade-offs have been demonstrated for pathogens infecting a single host species, it is unclear how they would impact the evolution of broad host-range pathogens that have transmission chains which routinely involve passages through multiple host species. It is also unclear how such trade-offs might impact the damage that pathogens inflict on short-lived cultivated crops; a situation where symptom intensities and host survival rates may have little impact on pathogen fitness. Here we address these unknowns by examining changes over the past ~110 years in the intensity of disease symptoms induced in maize by the broad host-range viral pathogen, maize streak virus (MSV). Specifically, we use the quantified symptom intensities displayed by differentially resistant maize genotypes

infected by cloned MSV isolates to phylogenetically infer the symptom intensities induced by ancestral MSV lineages following the emergence of MSV as a maize pathogen in the mid to late 1800s. Further, we verify the accuracy of these inferences using computationally-predicted ancestral MSV genomes that were then synthesised, made infectious and tested. We find that following the expansion of the MSV host-range to include maize, the intensity of MSV-induced symptom types that are indicative of harm to the host either remained constant (leaf stunting), or decreased (chloroplast destruction). Conversely, an increase was observed in chlorotic leaf areas, a symptom type that is indicative of how successfully MSV colonises the host cell populations upon which its insect transmission vectors. Therefore, despite the complication of MSV having a broad host-range, its apparent adaptation to a crop species with a short life span remains consistent with an evolutionary trade-off between the amount of harm inflicted on infected maize plants, and how effectively the virus positions itself within plants to enable onward transmission.